UNIVERSITY OF WISCONSIN-LA CROSSE

Graduate Studies

THE PACING LANDSCAPE: COMPARISON OF RELATIVE POWER OUTPUT IN HYPOXIA AND NORMOXIA

A Manuscript Style Thesis Submitted in Partial Fulfillment of the Requirements for the Degree of Master of Science, Clinical Exercise Physiology

Katherine R. Malterer

College of Science and Health
Clinical Exercise Physiology

December, 2013

THE PACING LANDSCAPE: COMPARISON OF RELATIVE POWER OUTPUT IN HYPOXIA AND NORMOXIA

By Katherine R. Malterer

We recommend acceptance of this thesis in partial fulfillment of the candidate's requirements for the degree of Master of Science, Clinical Exercise Physiology.

The candidate has completed the oral defense of the thesis.

Carl Foster, Ph.D. Thesis Committee Chairperson	5/22/13 Date
Glenn Wright, Ph.D. Thesis Committee Member	5/22/13 Date
Clayton Camic, Ph.D. Thesis Committee Member	5-22-13 Date

Thesis accepted

Steven Simpson, Ph.D. Graduate Studies Director	6/12/13 Date

ABSTRACT

Malterer, K.R. The pacing landscape: Comparison of relative power output in hypoxia and normoxia. MS in Clinical Exercise Physiology, December 2013, 56pp. (C. Foster)

Power output (PO) is systematically decreased at higher altitude; but, in air resisted activities the reduction in power losses to air friction is larger than the decreases in aerobic PO and result in a better performance (e.g. reduced time) at altitude. However, when comparing modeled races to actual world records, a performance deficit has been suggested. The purpose of this study was to determine if this underperformance could be attributable to a lower fractional utilization (average % PPO across the event) or errors of pacing strategy. Twelve healthy, well-trained cyclists performed two maximal incremental tests, two habituation trials, and two time trials (TT), one of each set in normoxia and one in hypoxia (F_IO_2 = 16%)(~2300m). When PO was analyzed as a percent of peak power output (%PPO) from the maximal tests in each respective condition (hypoxic TT compared with hypoxic maximal test and normoxic TT with normoxic maximal test), %PPO at the beginning of the trial tended to be greater in hypoxia, and correspondingly lower late in the trial, but with no significant difference in the average fractional utilization. This suggests that cyclists may use a detrimentally high initial power output at altitude.

ACKNOWLEDGEMENT

There are many, many people who deserve a word of thanks for helping me in the adventure that is a thesis. However, there are a few who are truly special and deserve to see it in writing.

I would like to thank Carl Foster, Clayton Camic, and Glenn Wright for advising me throughout my research and for being on my thesis committee. Also, thanks to Jos de Koning for your support from Amsterdam. Great minds do NOT think alike and I gained a new appreciation for academic collaboration this year. Thank you to all of the subjects who participated in this study. I am truly honored by your willingness to show me the very limits your athletic abilities. Thank you to Chris Dodge for being the master of all that is mechanical and such a bright spot in my day. I would like to also thank my family, friends, boyfriend, and LEHP brothers and sisters for all of the love and encouragement you flooded me with every day. A girl can't get any luckier than to have people like you in her life.

Finally, and most importantly, I need to thank Carl Foster. Without you, I would not be the person I am academically or personally. You provided me with innumerable opportunities and had unwavering faith that I would make the most of them. You are and will always be my most important mentor, my academic hero, and my friend. I only hope that I continue to make you proud in the years to come. Thank you, Carl.

TABLE OF CONTENTS

PAGE

LIST OF FIGURES

FIGURE PAGE

LIST OF APPENDICES

INTRODUCTION

Ambulatory, pursuit type sports such as running, swimming, cycling, and speed skating depend on a dynamic balance between power output from the muscles and power losses to the environment. Because much of muscular power output is dependent on aerobic metabolism, power output is systematically decreased at higher altitude. However, in air resisted activities such as cycling and speed skating, the reduction in power losses to air friction is larger than the decreases in aerobic power output (VO_{2max} and/or VO_2 at ventilatory/lactate threshold), with the result that performance is actually better (e.g. reduced time). However, recent observations (Heine, 2010) have suggested that world records set at altitude are not as good as they might be expected to be. This has prompted a reexamination of the elements of power production to determine whether systematic overestimation has occurred. Parameter estimates of VO_{2max} have been reexamined, with a slight downward revision (Wehrlin & Hallén, 2006). Gross mechanical efficiency is also slightly lower at altitude (Noordhof, Schoots, Hoekert, & de Koning, 2012), sufficient to account for about half the underperformance at altitude. Reasonable additional possibilities for underperformance at altitude are a lower sustainable relative power output, fractional utilization in the performance model of Joyner (Joyner & Coyle, 2008), or errors of pacing strategy.

Acute hypoxia, as seen at altitude, reduces heart rate maximum (HR_{max}) and maximum oxygen uptake (VO_{2max}) during peak maximal effort to compensate for lack of available oxygen and to prevent critical arterial desaturation (Mollard et al., 2007;

Noakes, Peltonen, & Rusko, 2001; Peltonen, Tikkanen, & Rusko, 2001). To maintain SaO_2 levels, a purposeful down regulation of power output (PO) is signaled by peripheral muscle fatigue and respiratory muscle fatigue (Amann et al., 2006; Amann, Pegelow, Jacques, & Dempsey, 2007; Millet, Muthalib, Jubeau, Laursen, & Nosaka, 2012; Noakes et al., 2001). This system is very sensitive, as shown by large decreases in PO with only small changes in inspired O_2 concentrations (Amann, 2011; Amann et al., 2006; Peltonen et al., 2001), even with no significant increases in the overall sensation of fatigue, blood lactate concentrations, and relative force output at the point of exhaustion compared to normoxia (Johnson et al., 2009; Romer et al., 2007).

In elite athletic performance of aerobic sports (e.g. running, skating, cycling), a higher VO_{2max} and lactate threshold allow for increased performance (Saltin & Astand, 1967). Altitude systematically reduces overall VO_{2max} (Amann et al., 2006) however, Costill, Thompson, and Roberts (1973) suggested VO_{2max} is not as important in predicting performance as the percentage of aerobic power ($\%VO_{2max}$) and $\%HR_{max}$ that an athlete can sustain during a given exercise bout. This has come to be known as 'fractional utilization', or the average percentage of VO_{2max} that can be sustained in events of different duration (Costill et al., 1973). If an athlete can perform at a higher $\%VO_{2max}$ with a lower % $lactate_{max}$ for a given workload then they would have the possibility of a greater power output attributable to aerobic energy production. Joyner (1991) took this concept to suggest a model for predicting the optimal marathon performance.

running speed = VO_{2max} x % VO_{2max} at lactate threshold x running economy

If aerobic energy is used efficiently with an optimal running economy (RE), running speed may be increased and performance improved, even without an increase in VO_{2max}. Joyner later adapted the model to a more general schematic of determinants of physiological factors of performance PO or velocity and included this component (Joyner & Coyle, 2008).

Performance velocity/PO = (Performance VO_2 (%VO_2 at LT x VO_{2max}) + Performance O_2 deficit (lactate buffering capacity + phosphagen depletion)) x Gross Mechanical Efficiency

Since cycling elicits very similar physiological needs as running, one of the few differences between cycling and running is that gross mechanical efficiency (GE), instead of RE, can be directly measured during cycling. When comparing efficiencies of running and cycling, GE can vary 20-30% in cyclists, whereas RE can vary up to 30-40% in runners (Joyner & Coyle, 2008). Efficiency then becomes an important deciding factor in performance. Altitude slightly reduces GE, possibly due a variety of factors such as increased metabolic work in hypoxia or increased cycling cadence (Clark et al., 2007; Noordhof et al., 2012).

However, reduction in GE does not fully account for the discrepancy between modeled and actual performance at moderate altitude (Noordhof et al., 2012). The effect of reduced aerobic power due to reduced VO_{2max} is less than that of the effect of reduced air density on PO loss, which should result in faster altitude specific results than have been recorded (Heine, 2010) (Figure 1). Since performance velocity is determined by fractional utilization of VO_{2max}, overall PO for a time trial (TT) would also only be a percent of the total Peak Power Output (%PPO) an athlete can reach. However, the

concept of fractional utilization assumes that the PO during an event is relatively constant. Studies over the last 20 years (summarized in Foster et al., 2012) indicate that the distribution of PO within an event (the pacing pattern) is: a) important to performance, and b) not constant, but distributed in highly characteristic ways depending on the duration of the event. If VO_{2max} is used at a paced submaximal percent to reach optimal performance, %PPO (as a derivative of $\%VO_{2max}$) would also be distributed in a paced pattern to achieve the best performance. This deciding factor would be visible in the athlete's pacing pattern.

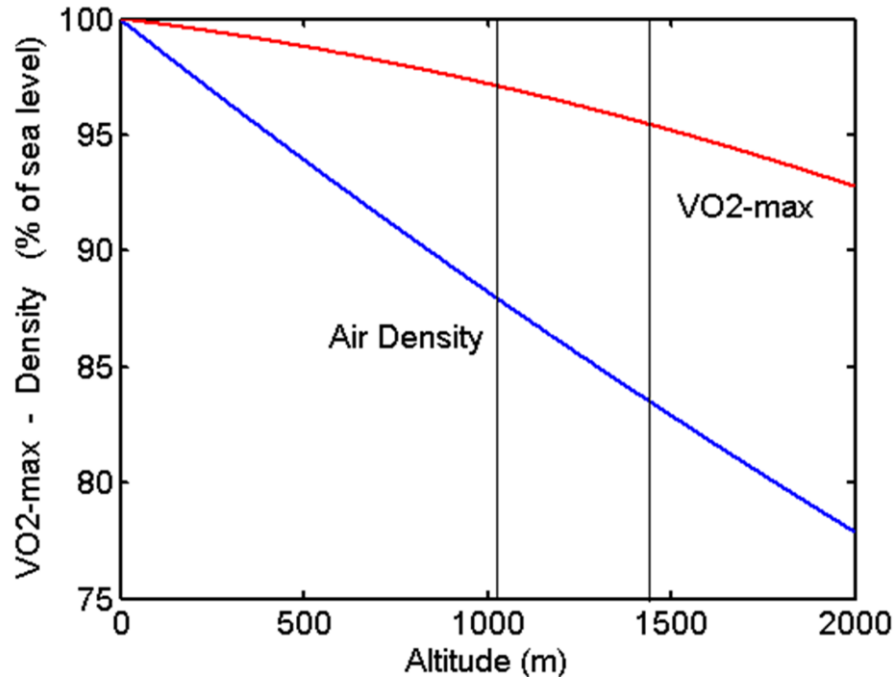

Figure 1. Decrease in Air Density in Relationship to Decreases in VO_{2max}. Note that the percent decrease in air density is larger than the decrease in VO_{2max}. The vertical reference lines represent the altitude of the Calgary (1030m) and Salt Lake City (1400m) speed skating ovals, at which every current world record has been set (Heine, 2010).

Pacing patterns may be defined as the distribution of muscular energetic output by continual modulation of PO to maximize performance and limit the magnitude of

homeostatic disturbance over a given distance, time, or task, based on a complex combination of peripheral and central feedforward/feedback mechanisms (Abbiss & Laursen, 2008**;** de Koning et al., 2011; Foster et al., 2012; Hill, Long, & Lupton, 1924; Noakes et al., 2001; Noakes, St. Clair Gibson, & Lambert, 2005; St. Clair Gibson et al., 2006). This continual 'internal conversation' is regulated by a "central governor" (CG) which detects disturbances in the body's homeostatic state via afferent input from the periphery (Amann et al., 2006) and sends efferent instructions to adjust PO to help the individual reach a defined endpoint while maintaining homeostatic disturbances within acceptable parameters (Abbiss & Laursen, 2008; Hill et al., 1924; Lambert, St. Clair Gibson, & Noakes, 2005; Noakes et al., 2001; St. Clair Gibson et al., 2006; Ulmer, 1996). The psychophysiological "language" of the CG is the rating of perceived exertion, or RPE (Borg, 1982) which has been correlated to increases in physiological changes such as muscular force, cardiopulmonary work, and blood lactate concentration (Albertus et al., 2005; Borg, 1982; Foster et al., 2009; Hampson et al., 2004; St. Clair Gibson et al., 2006) and is modulated by environmental challenges, such as altitude and thermal changes (Crewe, Tucker, & Noakes, 2008; St. Clair Gibson et al., 2006; Tucker, 2009). When presented with an unexpected challenge like hypoxia, RPE will continue to increase in a linear manner because reductions in PO are almost perfectly scaled to control the rate of change of homeostatic dusturbances (Johnson et al., 2009). The growth of RPE during any event, just like the CG's regulation of PO, seems to be scaled to the relative length of the event regardless of how different the length of the events are (Faulkner, Parfitt, & Roger, 2008; Joseph et al., 2008; Ulmer, 1996).

Several specific pacing patterns have been previously defined by (Abbiss & Larson, 2008) and include negative, positive, all out, and even pace. Statistical modeling of a theoretically perfect 1km and 4km cycling race has shown the optimal pace of these distances to consist of an "all out" (maximal momentary PO) start with a switch to Even Pace (where PO is held relatively constant) for the remainder of the event. However, observations of elite cyclists have shown an "endspurt" or "kick" as a consistent feature (de Koning, Bobbert, & Foster, 1999; Foster et al., 1993, Foster et al., 2012). This likely represents a reserve (Swart et al., 2009a; Swart et al., 2009b) of energy held by the CG as motor unit recruitment and metabolic reserves as a backup to prevent a catastrophic homeostatic disturbance in self-paced time trials (Crewe et al., 2008; Mauger, Jones, & Williams, 2011; St. Clair Gibson et al., 2006; Tucker, 2009) and prevent exhaustion before the completion of the race (Ulmer, 1996).

Initial selection of a pacing strategy, known as the pre-race template, is unconsciously based on the perceived distance or time to completion of the event as well as an expectation of effort required to reach either the expected endpoint (Tucker, 2009) or intermediate points within the event (Joseph et al., 2009). Once an event begins, internal and external homeostatic feedback appears to influence the CG and determine whether the pacing pattern remains the same or changes (Foster et al., 2009). This pre-exercise template has the ability to be "reset" or "hard-wired" based on previous experiences, training, and the knowledge of an endpoint (Abbiss & Laursen, 2008; Foster et al., 2009; Lambert et al., 2005; St. Clair Gibson et al., 2006; Ulmer, 1996), but is very resistant to change its initial selection of PO. Despite manipulations of warm-up O_2 conditions, similar power outputs in the beginning stages of time trials have been

observed regardless of whether the preceding warm-up was in normoxia or hypoxia (Henslin-Harris et al., 2013; Jaime et al., 2012). The selected pre-race template and initial power outputs continued to be for a TT pace in the familiar normoxic conditions of the sea level trained athletes despite hypoxic warm-up conditions which should have produced excessive afferent feedback and caused PO reduction by the CG (Johnson et al., 2009; Mollard et al., 2007; Peltonen et al., 2001; Tucker, 2009). This is significant as even small variations in pacing strategy during 1 and 4km TT can have substantial effects on performance (de Koning et al., 1999).

Thus, cycling performance at altitude is not only determined by the interaction of GE and the sustained PO from the aerobic and anaerobic energy systems, but it would be highly affected by pacing strategy (Clark et al., 2007). Hypoxia alone does not change pacing patterns as seen with PO, but it does decrease peak VO_2 (Clark et al., 2007) and GE (Noordhof et al., 2012). The pattern of sustained PO distribution would then be the key to unlocking why cycling performance at altitude is not as good as modeled. If the model (Joyner, 1991; Joyner & Coyle, 2008) is revisited with the important addition of pacing pattern included, a new model may look like the following:

Momentary PO = PPO (aerobic + anaerobic) x GE x Pacing (mean%PPO)

Aerobic and anaerobic energy, in the form of PPO, as well as a slight decrease in GE may be accounted for in hypoxic condition. However, there is still a deficit in performance that is not accounted for when compaired to what has been modeled, very likely explained by pacing patterns in the form of percent PPO. The purpose of this study is to determine if this remaining deficit may be due to a lower fractional utilization at altitude and the its pattern of distribution (pacing pattern). The CG and pre-race template

appear to adapt to changes in external environmental changes such as hypoxia, represented by internal homeostatic disturbances such as SaO_2, by adjusting PO. However, even in suboptimal conditions such as altitude, the pre-race template appears to have a delay in modifying itself to a more suitable PO. With a short distance such as a 4km TT, even a small miscalculation in initial pacing would be extremely detrimental to performance. Therefore, I hypothesize that despite sub-optimal conditions (hypoxia) the pre-race template will remain robust and cause individuals to have a greater initial %PPO, which potentially represents a sub-optimal pacing pattern for perfect performance. Also, if initial %PPO would be higher at altitude, more of the finite energy stores would be used earlier in a TT causing sustained and terminal PO's to be reduced.

METHODS

Subjects

The subjects were 12 healthy, well-trained cyclists and triathletes (Table 1). All subjects provided written informed consent, completed a health history questionnaire, and met current ACSM risk identification and stratification requirements for low risk. Subjects were asked to maintain their current training volume (3.5 hour average per week, as the study was conducted in the winter months) throughout the duration of testing and to refrain from exercise the day of a trial or vigorous exercise 24 hours prior to the trial. Participants were also asked to abstain from alcohol 24 hours prior to and caffeine on the days of testing. The testing protocol was approved by the University of Wisconsin – La Crosse Institutional Review Board for the Protection of Human Subjects. Hypoxic conditions were created to simulate the highest elevation of an Olympic competition (Mexico City, Mexico, ~ 2300m or 16.0% FiO_2 with normobaria).

Maximal Incremental Tests

Each subject performed two incremental exercise tests, one "maximal sea level" (MS) in normoxia and one "maximal altitude" (MA) in hypoxia, to determine VO_{2peak}, peak power output at VO_{2max} (PPO), and HR_{max} in both conditions. Testing was conducted on an electronically braked cycle ergometer (Lode Excaliber, Groningen, the Netherlands) with respiratory gas exchange measured using open circuit spirometry (AEI Technologies, Pittsburg, PA) and HR recorded with radiotelemetry (Polar Instruments, Port Washington, NY). SpO_2 was measured with a pulse oximeter (Allegiance Oxi-

Reader 2000, Allegiance Health Care, McGraw Park, IL). The incremental maximal testing protocols for both MS and MA consisted of the subject cycling at 25W for 3 minutes then increasing 25W every minute until exhaustion.

Table 1. Descriptive Physical Characteristics of Subjects and Incremental Test Values

Variable	Males (n = 9)	Females (n = 3)
Age (yrs)	30.2 ± 11.0	35.0 ± 14.9
Height (cm)	175.3 ± 7.4	162.2 ± 3.8
Weight (kg)	77.4 ± 8.2	64.0 ± 6.4
VO_{2peak} SL ($ml \cdot min^{-1} \cdot kg^{-1}$)	58.4 ± 7.4	42.7 ± 6.7
HR_{max} SL (bpm)	178.1 ± 16.2	178.7 ± 13.3
HR_{max} AL (bpm)	173.0 ± 15.7	176.7 ± 11.0
PPO SL (W)	344.4 ± 34.2	244.4 ± 38.7
PPO AL (W)	312.7 ± 26.5	222.2 ± 29.2

Mean ± Standard Deviation, SL = Sea Level, AL = Altitude

Familiarization and Time Trials

Following the maximal tests were two habituation 4km trials, with one at sea level (FS) and one at altitude (hypoxia) (FA) were performed to allow participants to become accustomed to the experimental protocol as well as the measurements taken, including the inspiratory resistance of the breathing apparatus, and use of the TT cycle ergometer (Veletron, Racermate, Seattle, WA). As a warm-up, participants cycled 5 minutes at 100W, 5 minutes at 50% PPO, 5 minutes at 75% PPO, 2 minutes at 100W, and 2 minutes at 25W (Figure 1). The altitude warm up was designed as a percentage of the altitude

specific PPO and the sea level warm up was designed as a percentage of the sea level specific PPO. Subjects reaching a PPO of less than 250W during either the MA or MS started warm-up at a PO of 50W instead of 100W for the designated 5 minute and 2 minute stages to prevent undue fatigue before the trial. During the TT, blood samples for lactate concentrations [HLa] were taken at 0.0 km, 2.0 km, and 4.0 km. RPE and SpO_2 were recorded every 0.4 km. Immediately following the TT the subject performed a 7 minute cool down with 2 minutes at 25W and 10 minutes at 50% of either the altitude or sea level specific maximal incremental test PPO. Power output measurements during the time trial were made every 0.37s by the Velotron ergometer, and subsequently averaged over every 0.4 km distance for analysis.

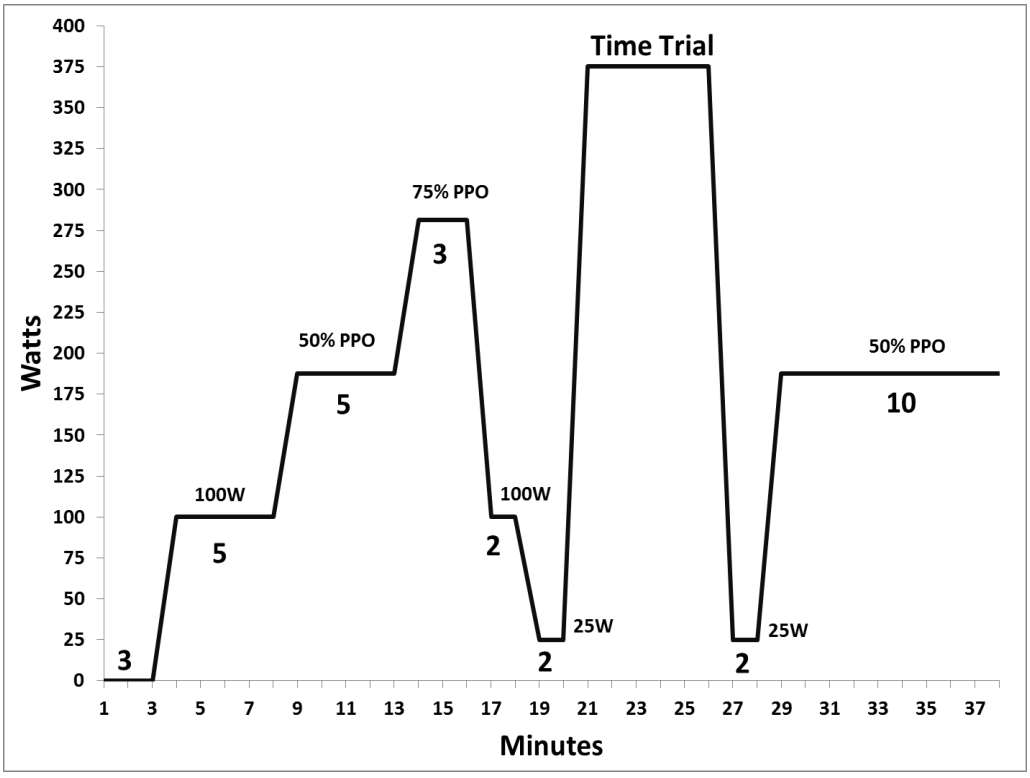

Figure 2. Time Trial Protocol with Warm-up and Cool Down. The %PO measurements during each trial were normalized to the altitude specific PVO_{2max} observed during the incremental tests.

Statistical Analysis

Statistical analysis was performed on results of power output, altitude normalized percent power output, lactate, rating of perceived exertion, saturation, and heart rate using repeated measures ANOVA to compare differences between altitude and sea level at each .4km. Differences were also compared between each successive .4km in to determine effects due to increasing distance. All differences were both absolute and normalized to the PO during the altitude specific incremental test. A $p < 0.05$ was accepted as statistically significant. When ANOVA was not necessary, mean values were compared using paired t-tests. Mean values determining differences between altitude and sea level conditions were analyzed for power output, altitude normalized percent power output, performance time, blood lactate, rating of perceived exertion, saturation, and heart rate. All analyses were conducted using the Statistical Package for the Social Sciences (SPSS, Version 19; SPSS Inc., Chicago, IL.)

RESULTS

The total average PO for the altitude TT (241.64 ± 49.42 W) was significantly lower ($p \leq 0.001$) than sea level (270.64 ± 55.82 W) and changed significantly ($p \leq 0.001$) over the distance (from start to end) of the TT although there was no significant interaction between distance and altitude ($p = 0.239$) (Figure 3). The pattern of PO was the same in both TT, with the typical U shaped pattern seen in events of this duration (Foster et al., 2012). Percent PPO showed a trend ($p = 0.072$) of a higher value at altitude at the start and a lower value at the finish, but was not significant ($p = 0.238$). However, percent PPO did change over distance ($p \leq 0.001$) (Figure 4). The time that it took to complete the altitude TT (409.83 ± 36.71 sec) was significantly ($p \leq 0.001$) longer than sea level (392.08 ± 35.53 sec) (Figure 5). Blood lactate concentration significantly increased ($p \leq 0.001$) over the length of the TT, but was not significantly different ($p = 0.462$) between altitude and sea level (SL = 5.1±1.6, 11.0±2.3, 13.4±2.5 $mmol \cdot L^{-1}$; AL = 5.47±1.76, 11.0±1.9, 14.2±2.0 $mmol \cdot L^{-1}$) with no significant interaction ($p = 0.414$) between distance and altitude (Figure 6). RPE significantly increased ($p = 0.004$) over the TT length (Figure 7), but was not significantly different ($p = 0.384$) between sea level and altitude with no significant interaction ($p = 0.760$) between distance and altitude (Table 2). Oxygen saturation was significantly lower ($p \leq 0.001$) at altitude and increased significantly ($p \leq 0.001$) over the length of the TT (Table 2), but without significant interaction ($p = 0.620$) between altitude and distance (Figure 8). Heart rates increased

significantly over the length of the TT ($p \leq 0.001$) (Figure 9) and with altitude ($p = 0.018$) (Table 2) with a significant interaction ($p = 0.017$) between altitude and TT length.

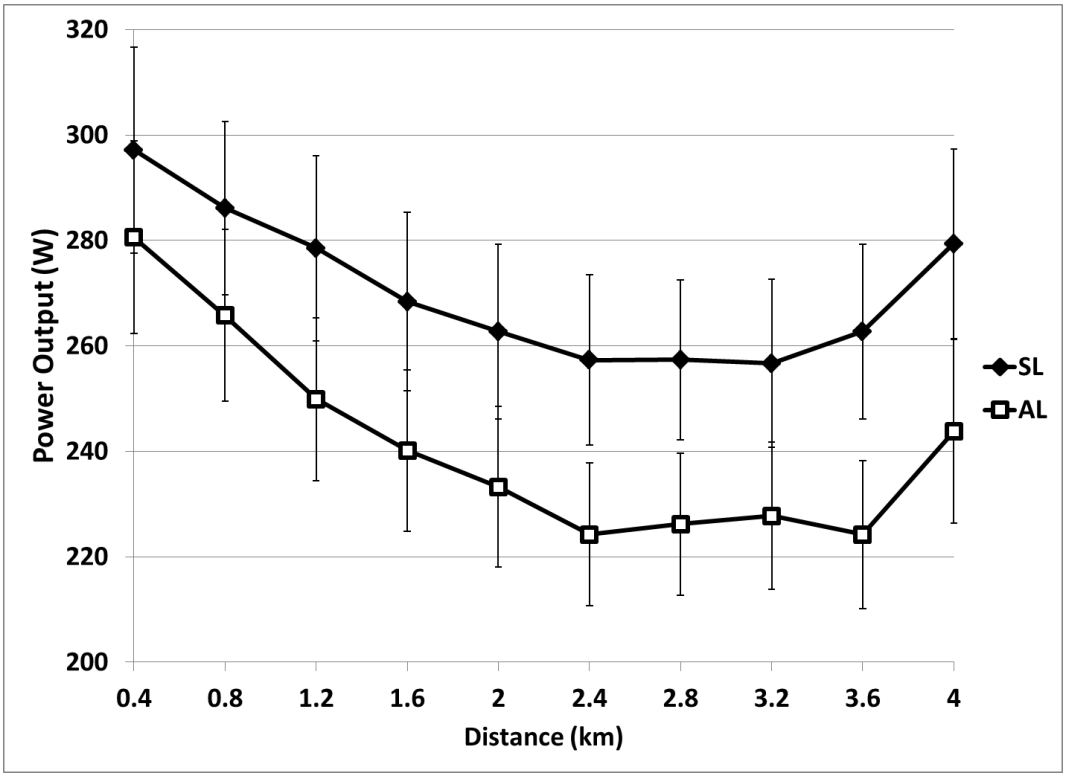

Figure 3. Power Output. Average PO for the TT was lower at altitude and changed with increasing distance.

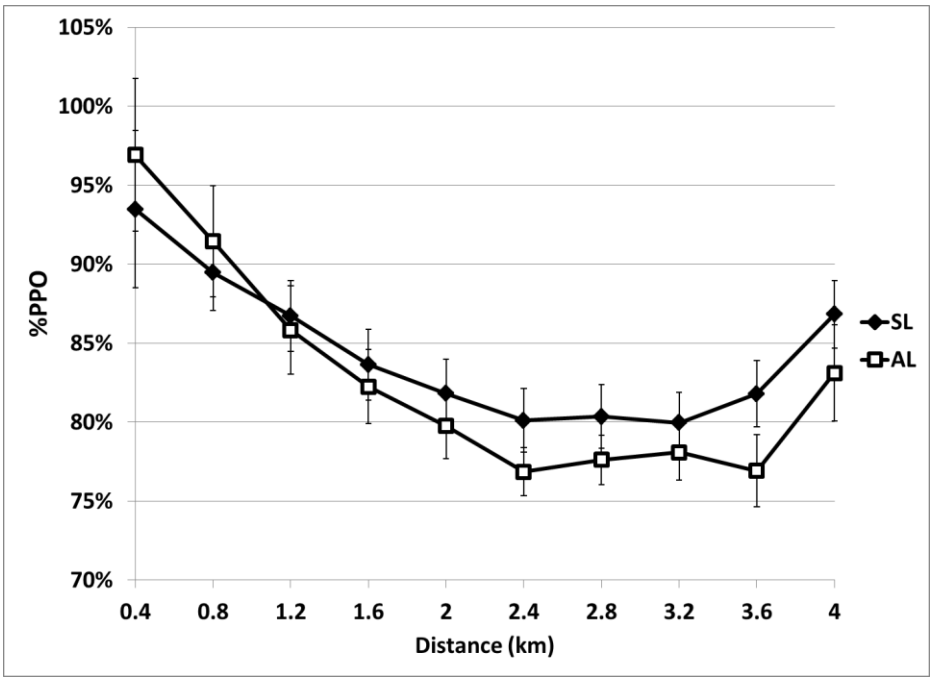

Figure 4. Altitude Normalized Percent Power Output. A trend was evident for percent PPO to start higher and finish lower during the altitude TT, but mean percent PPO was not significantly different between altitude and sea level. However, %PPO changed over the length of the TT.

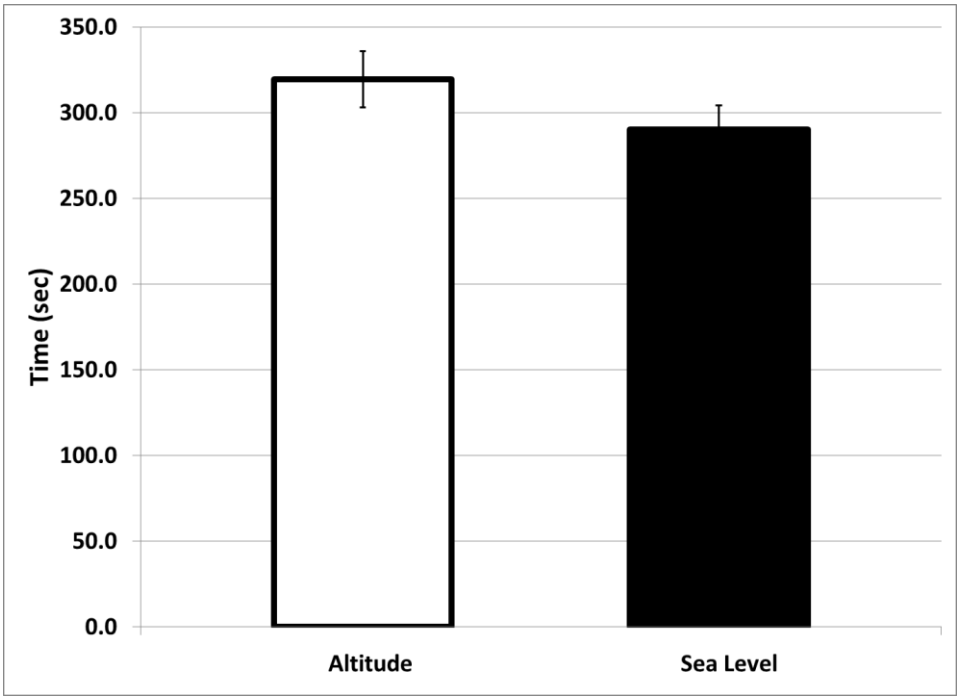

Figure 5. Performance Time. Cycling time was increased with altitude.

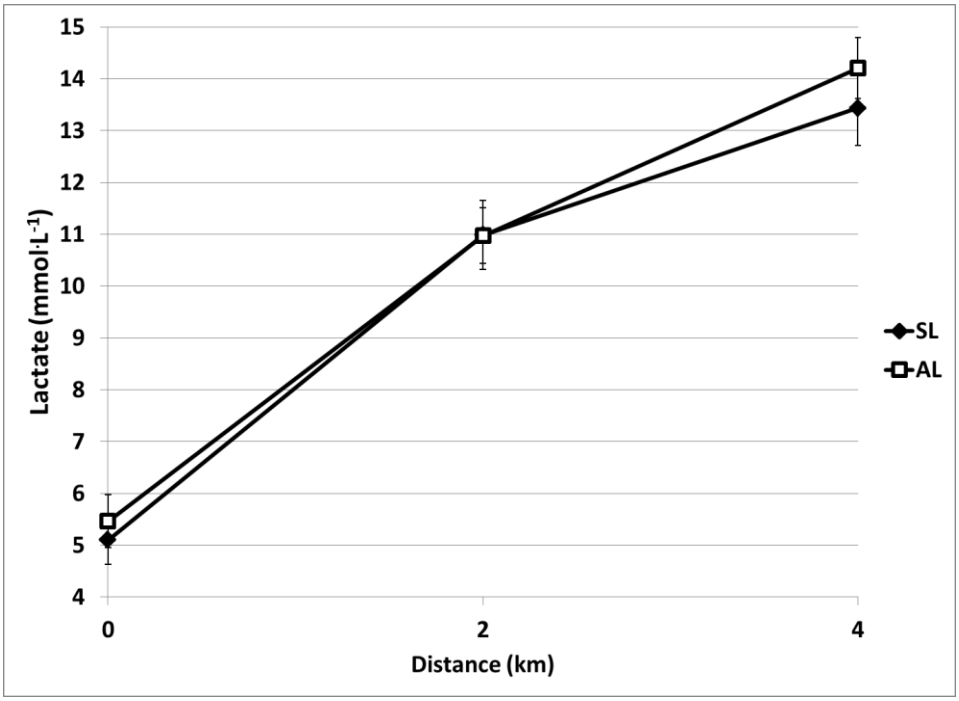

Figure 6. Blood Lactate. Blood lactate increased with distance, but was not different between altitude and sea level.

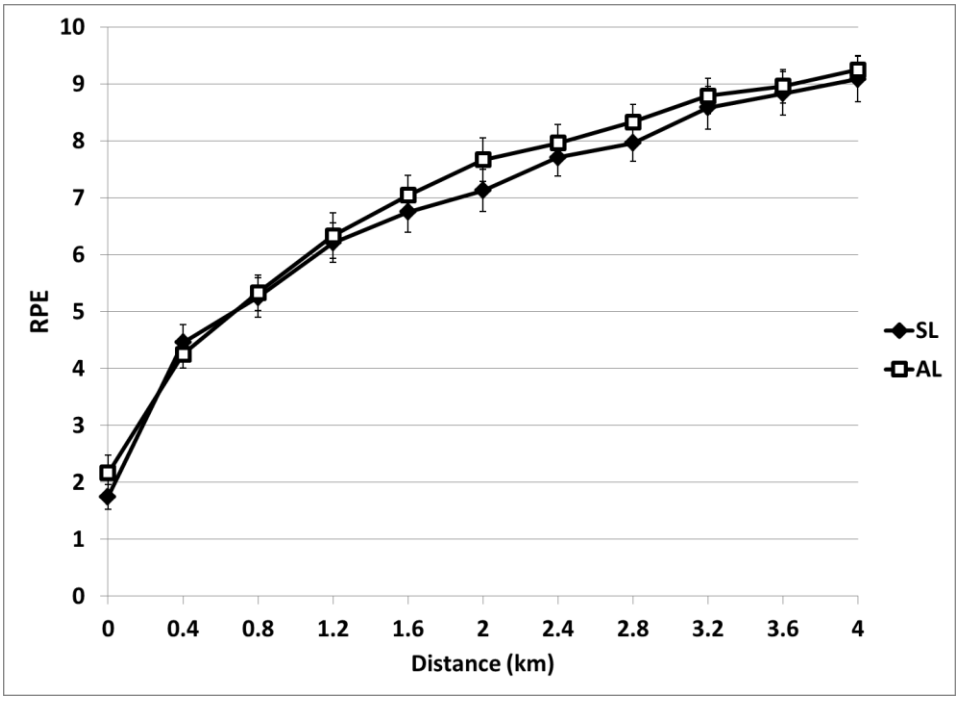

Figure 7. Rating of Perceived Exertion. RPE growth was not different between altitude and sea level, but did increase with distance.

Table 2. Mean Rating of Perceived Exertion, Saturation, and Heart Rate Results

	Distance (km)	Sea Level	Altitude
RPE (1-10)	.4	4.5 ± 1.1	4.3 ± .84
	.8	5.3 ± 1.2	5.3 ± 1.1
	1.2	6.2 ± 1.2	6.3 ± 1.4
	1.6	6.8 ± 1.2	7.0 ± 1.2
	2.0	7.1 ± 1.3	7.7 ± 1.3
	2.4	7.7 ± 1.1	9.0 ± 1.1
	2.8	8.0 ± 1.1	8.3 ± 1.1
	3.2	8.6 ± 1.3	8.8 ± 1.1
	3.6	8.8 ± 1.3	9.0 ± 1.0
	4.0	9.1 ± 1.4	9.3 ± .84
Saturations (%)	.4	98 ± 1	91 ± 4
	.8	96 ± 2	88 ± 4
	1.2	95 ± 3	89 ± 4
	1.6	96 ± 1	88 ± 3
	2.0	95 ± 3	88 ± 4
	2.4	94 ± 3	87 ± 4
	2.8	94 ± 4	86 ± 4
	3.2	94 ± 3	85 ± 4
	3.6	93 ± 4	85 ± 5
	4.0	93 ± 4	85 ± 5
Heart Rate (bpm)	.4	148 ± 17	145 ± 16
	.8	163 ± 15	163 ± 14
	1.2	169 ± 14	167 ± 14
	1.6	171 ± 14	169 ± 14
	2.0	173 ± 14	169 ± 14
	2.4	173 ± 15	169 ± 15
	2.8	174 ± 14	170 ± 15
	3.2	175 ± 14	172 ± 14
	3.6	177 ± 14	173 ± 14
	4.0	197 ± 14	174 ± 14

Means ± Standard Deviation

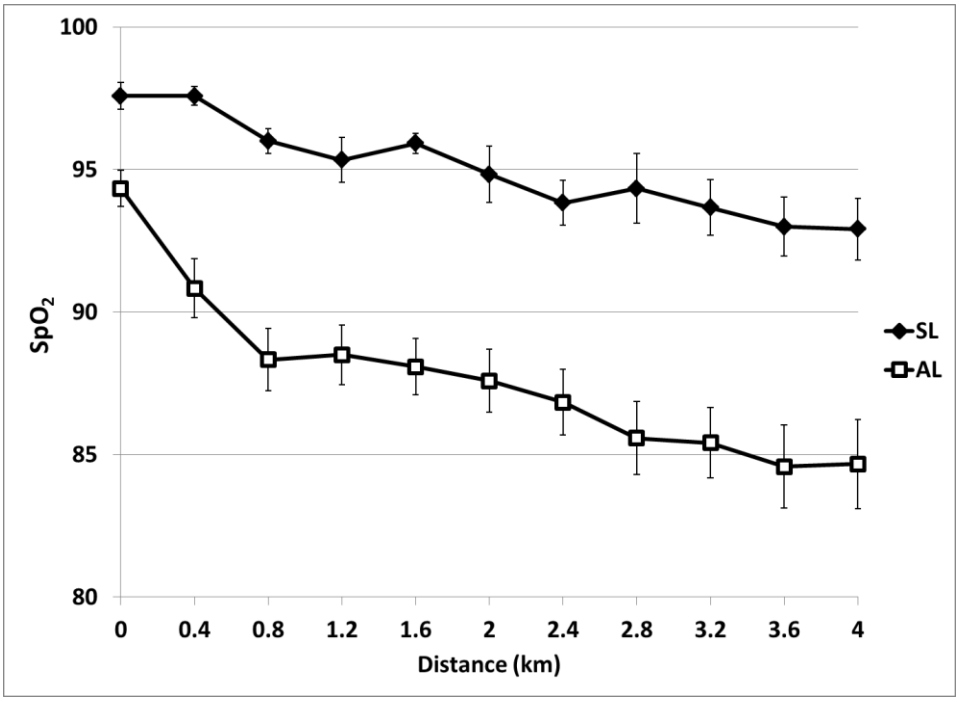

Figure 8. Saturation. Saturations decreased with increasing distance and were lower at altitude.

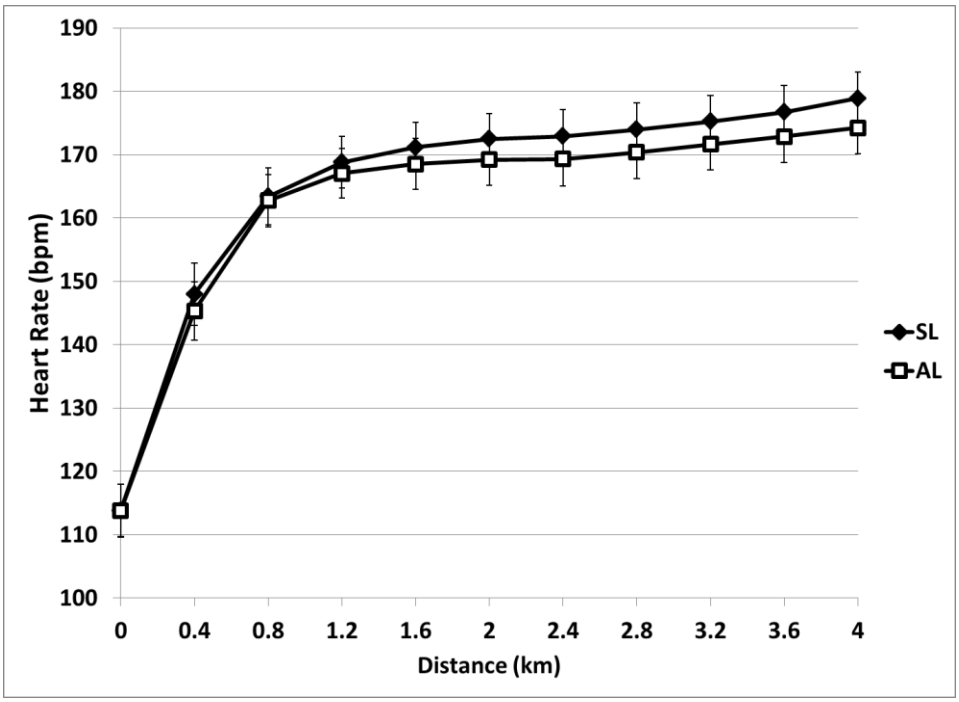

Figure 9. Heart Rate. Heart rates increased with increasing distance and were lower at altitude. An interaction between altitude and distance was also evident.

DISCUSSION

Previous studies (Henslin-Harris et al., 2013; Jaime et al., 2012) indicated that initial PO in hypoxic conditions was similar to sea level conditions due to an apparent robustness of the pre-race template even in the face of pre-exercise hypoxia (Henslin-Harris et al., 2013) or a perceptually harder warm-up caused by hypoxia (Jaime et al., 2012). The current study suggests that when PO is normalized to a maximal incremental test in the same normoxic-hypoxic conditions, a trend may be visible for a greater percent PPO at the start in hypoxic conditions representing an important difference in pacing patterns which would agree with the findings of Jaime et al. (2012).

Power output during the 4km TT was significantly affected by both hypoxia and increasing distance as was expected (Foster et al., 2012; Henslin-Harris et al., 2013; Jaime et al., 2012; Johnson et al., 2009) and was overall lower in the hypoxic condition. However, the shape of both PO curves was the same as has been seen other 4km time trials and followed the same general pattern that is seen with elite athletes (Foster et al., 2012). When PO was analyzed as %PPO in each respective conditions (hypoxic TT compared with hypoxic maximal test and normoxic TT with normoxic maximal test) the difference is not significant. %PPO at the beginning of the trial tended to be greater in hypoxia, and correspondingly lower late in the trial; however, the mean %PPO over the entire length of the trial was not different. This may be due to the short distance of the 4km. As a TT progresses, finite stores of energy from the aerobic and anaerobic systems are utilized. It is suggested that it is the pattern of utilization from the CG that determines

the PO at each moment. If the TT of this study were longer, a more significant decrease in percent PPO may have been seen if there was indeed a significantly larger initial %PPO at altitude. More of the stores would be used, leaving a larger deficit with which to complete the rest of the TT with. This trend of %PPO starting higher and finishing lower in competitions at altitude can also be identified in World Championship speed skating races as well (McClennen, 2013).

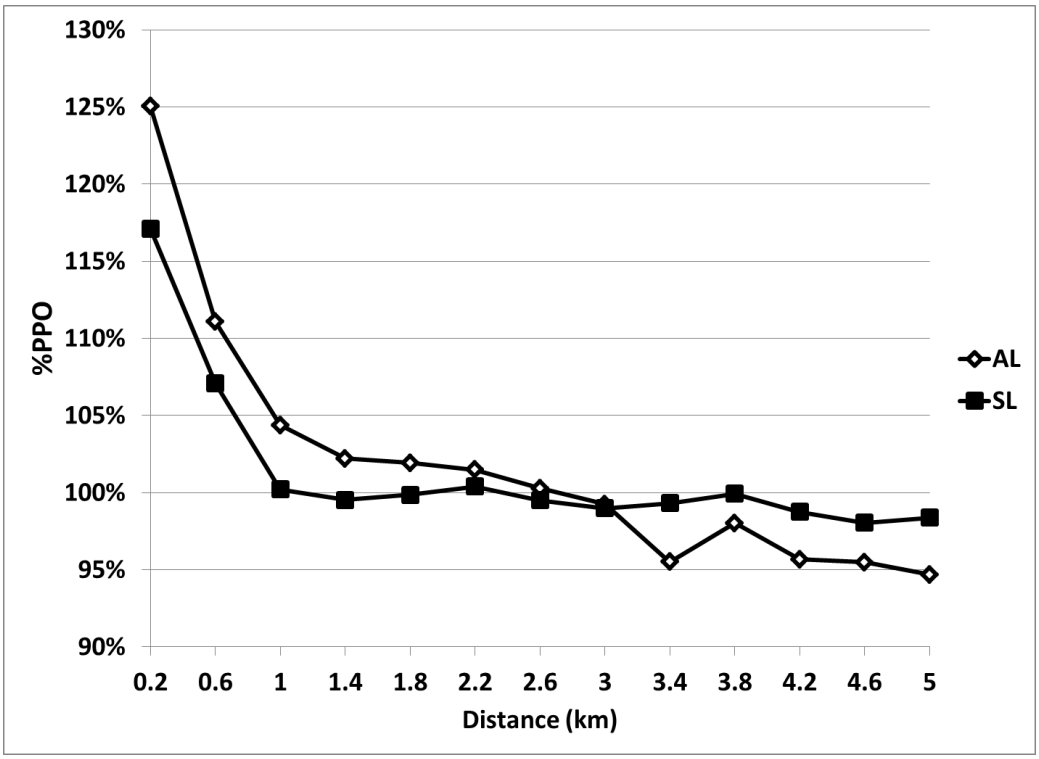

Figure 10. Modeled %PPO ISU World Championship Results. Mean modeled percent peak power output during 5km International Skating Union (ISU) speed skating races performed during World Cup (AL) and World Single Distance Championships (SL). The overall pattern is strikingly similar to the experimental results observed in this study (Figure 4).

Both HR and SpO_2 were significantly lower in hypoxia compared to normoxia with both of these factors being affected by increased distance. This supports the CG theory that the body down-regulates cardiovascular responses to prevent catastrophic

desaturation. A decreased HR_{max} would also contribute to a lower VO_{2peak} as suggested by Clark et al. (2007) and reduced aerobically attributable energy production, reflective in the lower mean PO at altitude.

Lactate was not significantly different between both conditions despite a lower PO in hypoxia. This may be due to lower VO_{2peak} and higher contribution from the anaerobic system in response to the hypoxia with the CG moderating PO to prevent lactate levels from exceeding a catastrophic limit before the endpoint of the race. Since lactate is an indicator of anaerobic work it is therefore an indicator of the PPO portion of our revised Joyner Model.

RPE increased significantly with distance and was not different between altitude and sea level, similar to results found by Johnson et al. (2009), Henslin-Harris et al. (2013), and Jaime et al. (2012) and continues to support the CG theory which predicts that PO will be reduced so that the individual does not reach a critical level of homeostatic disturbance before completion of the event.

The adjustment of %PPO, combined with a constant GE, and a definable PPO could explain the variations in momentary PO changes. Hypoxia would cause a lower PPO for a given event. The individual then would have that finite energy with which to finish the race adjusting it as %PPO to generate a moment to moment PO.

A possible limitation to this study would include the limited size to the testing group. The availability of an "elite" pool of cyclists was limited by the unwillingness of elite athletes to undergo extensive laboratory testing. Sub-elite, but experienced and habituated subjects were recruited instead. Another limitation could be the learning effect of performing short distance (4km) time trials with primarily endurance trained athletes.

Pacing strategies might continue to evolve is subjects were given more practice sessions before the final TT's (Foster et al., 2009), however, final maximal habituated performance would still not be as good as it would be if sprint cyclists were recruited.

In conclusion, no significant differences in total %PPO or its pacing pattern were seen between acute hypoxic and sea level cycling TT's. However, a trend was indicated for a higher %PPO at the start of hypoxic conditions which may lead us to investigate further into fractional utilization as the cause of underperformance at altitude. A revised version of Joyner's Model (Joyner & Coyle, 2008) with an included pacing component might yield more accurate models of cycling in hypoxia. Also, a revision of Heine's model of cycling (Heine, 2010) with the inclusion of a fractional utilization factor might also help account for the underperformance of cyclists at altitude.

REFERENCES

Abbiss, C. R., & Laursen, P. B. (2008). Describing and understanding pacing strategies during athletic competition. *Sports Medicine*, 38(3), 239-252.

Albertus, Y., Tucker, R., St. Clair Gibson, A., Lambert, E. V., Hampson, D. B., & Noakes, T. D. (2005). Effect of distance feedback on pacing strategy and perceived exertion during cycling. *Medicine and Science in Sports and Exercise*, 37(3), 461-468.

Amann, M. (2011). Central and peripheral fatigue: Interaction during cycling exercise in humans. *Medicine and Science in Sports and Exercise*, 43(11), 2039-2045.

Amann, M., Eldridge, M. W., Lovering, A. T., Stickland, M. K., Pegelow, D. F., & Dempsey, J. A. (2006). Arterial oxygenation influences central motor output and exercise performance via effects on peripheral locomotor muscle fatigue in humans. *Journal of Physiology*, 573(3), 937-952.

Amann, M., Pegelow, D. F., Jacques, A. J., & Dempsey, J. A. (2007). Inspiratory muscle work in acute hypoxia influences locomotor muscle fatigue and exercise performance of healthy human beings. *American Journal of Physiology: Regulatory, Integrative and Comparative Physiology*, 293, R2036-R204.

Borg, G. A. U. (1982). Psychophysical bases of perceived exertion. *Medicine and Science in Sports and Exercise*, 14(5), 377-381.

Clark, S. A., Bourdon, P. C., Schmidt, W., Singh, B., Cable, G., Onus, K. J.,… Aughey, R. J. (2007). The effect of acute simulated moderate altitude on power, performance and pacing strategies in well-trained cyclists, *European Journal of Applied Physiology*, 102, 45-55.

Costill, D. L., Thompson, H., & Roberts, E. (1973). Fractional utilization of the aerobic capacity during distance running, *Medicine and Science in Sports*, 5(4), 248-252.

Crewe, H., Tucker, R., & Noakes, T. D. (2008). The rate of increase in rating of perceived exertion predicts the duration of exercise to fatigue at a fixed power output in different environmental conditions. *European Journal of Applied Physiology*, 103, 569-577.

de Koning, J. J. (2010). World Records: How much athlete? How much technology?. *International Journal of Sports Physiology and Performance*, 5, 262-267.

de Koning, J. J., Bobbert, M. F., & Foster, C. (1999). Determination of optimal pacing strategy in track cycling with an energy flow model. *Journal of Science and Medicine in Sport*, 2(3), 266-277.

de Koning, J. J., Foster C., Bakkum A., Kloppenburg S., Thiel C., Joseph T.,... Porcari J. P. (2011). Regulation of pacing strategy during athletic competition. *PLoS ONE*, 6(1), 1-5.

Faulkner, J., Parfitt, G., & Roger, E. (2008). The rating of perceived exertion during competitive running scales with time. *Psychophysiology*, 45(6), 977-985.

Foster, C., de Koning, J. J., Bischel, S., Casolino, E., Malterer, K., OBrien, K.,... Van Tunen, J. (2012). Pacing strategies for endurance performance. In I. Mujika (Ed.), *Endurance training science and practice* (1 ed., Vol. 1, pp. 85-98). Trapagaran, Bizkaia: Samper Impresores.

Foster, C., Hendrickson, K. J., Peyer, K., Reiner, B., de Koning, J. J., Lucia, A.,… Wright, G. (2009). Pattern of developing the performance template. *British Journal of Sports Medicine*, 43, 765-769.

Foster, C., Snyder, A. C., Thompson, N. N., Green, M. A., Foley, M., & Schrager, M. (1993). Effect of pacing strategy on cycle time trial performance. *Medicine and Science in Sports and Exercise*, 25(3), 383-388.

Hampson, D. B., St. Clair Gibson, A., Lambert, M. I., Dugas, J. P., Lambert, E. V., & Noakes, T. D. (2004). Deception and perceived exertion during high intensity running bouts. *Perceptual and Motor Skills*, 98, 1027-1038.

Heine, M. (2010). Improved speed skating performance at altitude is overestimated by energy flow model: 2885: Board# 236 June 4 2: 00 pm-3: 30 pm. *Medicine and Science in Sports and Exercise*, 42(5), 784.

Henslin-Harris, K. B., Foster, C., de Koning, J. J., Dodge, C., Wright, G. A., & Porcari, J. P. (2013). Rapidity of response to hypoxic conditions during exercise. *International Journal of Sports Physiology and Performance*, 8, 330-335.

Hill, A. V., Long, C. N. H., & Lupton, H. (1924). Muscular exercise, lactic acid and the supply and utilization of oxygen. *Proceedings of the Royal Society: Biological Sciences*, 97, 155-176.

Jaime, C., Nyberg, K., Condello, G., Casolino, E., Dodge, C., Marroyo-Rodriguez, J.,… Foster, C. (2012). *The effect of manipulating the pre-exercise template on time trial performance*. In Meeusen, R., Duchateau, J., Roelands, B., Klass, M., De Geus, B., Baudry, S., Tsolakidis, E. (Eds.), *17th annual congress of the European college of sport science book of abstracts* (p. 74).

Johnson, B. D., Joseph, T., Wright, G., Battista, R. A., Dodge, C., Balweg, A.,… Foster, C. (2009). Rapidity of responding to a hypoxic challenge during exercise. *European Journal of Applied Physiology*, 106, 493-499.

Joseph, T., Johnson, B., Battista, R. A., Wright, G., Dodge, C., Porcari, J. P.,… Foster, C. (2008). Perception of fatigue during simulated competition. *Medicine and Science in Sport and Exercise*, 40(2), 381-386.

Joyner, M. J. & Coyle, E. F. (2008). Endurance exercise performance: The physiology of champions. *Journal of Physiology*, 586(1), 35-44.

Joyner, M. J. (1991). Modeling: optimal marathon performance on the basis of physiological factors. *Journal of Applied Physiology*, 70(2), 683-687.

Lambert, E. V., St. Clair Gibson, A., & Noakes, T. D. (2005). Complex systems model of fatigue: integrative homeostatic control of peripheral physiological systems during exercise in humans. *British Journal of Sports Medicine*, 39, 52-62.

Mauger, A. R., Jones, A. M., & Williams, C. A. (2011). The effect of non-contingent and accurate performance feedback on pacing and time trial performance in 4-km track cycling. *British Journal of Sports Medicine,* 45(3), 225-229.

McClennan, J. (2013). World records. Speedskatingresults.com. Retrieved May 1, 2013, from http://www.speedskatingresults.com/index.php?p=9.

Millet, F. Y., Muthalib, M., Jubeau, M., Laursen, P. B., & Nosaka, K. (2012). Server hypoxia affects exercise performance independently of afferent feedback and peripheral fatigue. *Journal of Applied Physiology*, 112, 1335-1344.

Mollard, P., Woorons, X., Letournel, M., Lamberto, C., Favret, F., Pichon, A.,… Richalet, J. (2007). Determinants of maximal oxygen uptake in moderate acute hypoxia in endurance athletes. *European Journal of Applied Physiology*, 100, 663-673.

Noakes, T. D., Peltonen, J. E., & Rusko, H. K. (2001). Evidence that a central governor regulates exercise performance during acute hypoxia and hyperoxia. *Journal of Experimental Biology,* 204, 3225-3234.

Noakes, T. D., St. Clair Gibson, A., & Lambert, E.V. (2005). From catastrophe to complexity: A novel model of integrative central neural regulation of effort and fatigue during exercise in humans: Summary and conclusions. *British Journal of Sports Medicine,* 39, 120-124.

Noordhof, D. A., Schoots, T., Hoekert, D. H., & de Koning, J. J. (2012). Is gross efficiency lower at acute simulated altitude than at sea level?. *International Journal of Sports Physiology and Performance*, 7, 319-322.

Peltonen, J. E., Tikkanen, H. O., & Rusko, H. K. (2001). Cardiorespiratory responses to exercise in acute hypoxia, hyperoxia, and normoxia. *European Journal of Applied Physiology*, 85, 82-88.

Romer, L. M., Haverkamp, H. C., Amann, M., Lovering, A. T., Pegelow, D. F., & Dempsey, J. A. (2007). Effect of acute severe hypoxia on peripheral fatigue and endurance capacity in healthy humans. *American Journal of Physiology: Regulatory, Integrative and Comparative Physiology*, 292, R598-R606.

Saltin, B. & Astrand, P. (1967). Maximal oxygen uptake in athletes[1], *Journal of Applied Physiology*, 23(3), 353-358.

St. Clair Gibson, A., Lambert, E. V., Rauch, L. H. G., Tucker, R., Baden, D. A., Foster, C., & Noakes, T. D. (2006). The role of information processing between the brain and peripheral physiological systems in pacing and perception of effort. *Sports Medicine*, 36(8), 705-722.

Swart, J., Lamberts, R. P., Lambert, M. I., Gibson, A. S. C., Lambert, E. V., Skowno, J., & Noakes, T. D. (20092). Exercising with reserve: Evidence that the central nervous system regulates prolonged exercise performance. *British Journal of Sports Medicine*, 43(10), 782-788.

Swart, J., Lamberts, R. P., Lambert, M. I., Lambert E. V., Woolrich, R. W., Johnston, S., & Noakes, T. D. (2009b). Exercising with reserve: Exercise regulation by perceived exertion in relation to duration of exercise and knowledge of endpoint. *British Journal of Sports Medicine*, 43(10), 775-781

Tucker, R. (2009). The anticipatory regulation of performance: the physiological basis for pacing strategies and the development of a perception-based model for exercise performance. *British Journal of Sports Medicine*, 43(6), 392-400.

Ulmer, H. V. (1996). Concept of an extracellular regulation of muscular metabolic rate during heavy exercise in humans by psychophysiological feedback. *Experientia*, 52, 416-420.

Wehrlin, J. P., & Hallén, J. (2006). Linear decrease in VO_{2max} and performance with increasing altitude in endurance athletes. *European Journal of Applied Physiology*, 96(4), 404-412.

APPENDIX A

PHYSICAL ACTIVITY READINESS QUESTIONNAIRE (PAR-Q)

Physical Activity Readiness Questionnaire (PAR-Q)

Name:_____ Age:_____ Date:_____
Local Address:_____
Telephone:_____ E-mail:_____

Read each question carefully and place an "**X**" in on the appropriate line. Please answer honestly and to the best of your knowledge.

If you answered yes:
If you answered yes to one or more questions, consult a physician before partaking in

Yes	No	
		1. Has a doctor ever said that you have a heart condition **and** that you should only do physical activity recommended by a doctor?
		2. Do you feel pain in your chest when you do physical activity?
		3. In the past month, have you had chest pain when you were not doing physical activity?
		4. Do you lose your balance because of dizziness or do you ever lose consciousness?
		5. Do you have a bone or joint problem that could be made worse by a change in you physical activity?
		6. Is your doctor currently prescribing drugs (for example, water pills) for your blood pressure or heart condition?
		7. Do you know of **any other reason** why you should not do physical activity?

this study. You should ask for a medical clearance along with information about specific exercise limitations you may have.

If you answered no:
If you answered no to all the PAR-Q questions, you can be reasonably sure that you can exercise safely and have low risk of having any medical complications from exercise.

Participant _____ Signature _____ Date _____

Researcher _____ Signature _____ Date _____

APPENDIX B

INFORMED CONSENT

INFORMED CONSENT

"The Pacing Landscape: Comparison of Relative Power Output in Hypoxia and Normoxia"

Principle Investigator: Katherine Malterer
E-mail: malterer.kath@uwlax.edu
Address: 621 Jackson St. Apt 6.
La Crosse, WI 54601
Telephone: 507-420-5733

Faculty Advisor: Carl Foster, PhD
E-mail: cfoster@uwlax.edu
Address: 133 Mitchell Hall, UW-L
La Crosse, WI 54601
Telephone: 608-785-8687

1) I,_____, give my informed consent to participate in this study designed to evaluate if altitude (hypoxia) will change the overall, relative power output of athletes in a time trial setting when compared to sea level performance. I also consent to the presentation, publication, and other release of summary data from the study which is not identifiable with myself. I have been informed that:
    a. All tests will take place in the Human Performance Lab of Mitchell Hall (room 225)
    b. This study will require me to perform 6 tests, 3 in a hypoxic (low oxygen) state and 3 in normoxic (normal oxygen level) state. Of these 6 tests, 2 will be maximal incremental protocols, 2 will be 4km habituation trials, and 2 will be 4km time trials with
    c. Each test lasting between 30 -60 minutes with a total time commitment of 6 hours of testing within a 4 week period.
    d. During all efforts, my performance will be measured quantitatively by the amount of time it takes to complete each time trial, heart rate, blood lactate levels, ventilatory markers, arterial oxygen saturation, and power output as recorded by a chest heart rate monitor, finger prick, snorkel-like breathe analyzer, finger monitor, and cycle ergometer.
2) I understand that there are no foreseeable risks for this experiment other than fatigue and slight discomfort from blood lactate sampling and air resistance/dryness of the breath analyzer.
3) I have been informed that there are no primary benefits to myself other than knowledge of my maximal capacities and time trial performances.
4) I have been informed that the investigator will answer any and all questions I have regarding the procedures throughout the entire course of the study.
5) I have been informed that all information will be kept confidential through the use of number codes and my data will not be linked to any personally identifiable information.
6) I have been informed that I am free to decline to participate or to withdraw from the study at any time without penalty.

Questions regarding study procedures may be directed to the principle investigator, Katherine Malterer, or to the faculty advisor, Dr. Carl Foster. Questions regarding the protection of human subjects may be directed to the UW-La Crosse Institutional Review Board for the Protection of Human Subjects (608-785-8007 or irb@uwlx.edu).

Participant _____ Signature _____ Date _____

Researcher _____ Signature _____ Date _____

APPENDIX C

PRE-PARTICIPATION GUIDELINES

Thank you for choosing to participate in my thesis study of "The Pacing Landscape: A Comparison of Relative Power Output at Altitude and Sea Level". I really appreciate it!

This study will require 6 total tests consisting of 2 incremental maximal tests, 2 familiarization trials, and 2 time trials. One of each test will be completed at a simulated altitude of 8,000ft and the other at standard La Crosse altitude of 670ft. Incremental maximal tests should take about 30 minutes, but familiarization and time trial tests may take up to an hour. Blood lactate from a finger prick will be taken along with heart rate and gas exchange for all tests.

Just a few things to note BEFORE coming in for EVERY testing day:

- Please abstain from any caffeine on the day of your test prior to coming in to cycle.
- Please be well hydrated and rested before your testing day.
- Please abstain from significant alcohol consumption at least 24 hours before testing.
- Please be sure to eat at an appropriate time before your test to ensure that you are able to perform at your best.
- Please do not participate in any other workouts on the same day prior to your test as we are looking for maximal efforts on all trials.
- The day before a test, you may do a light workout just as you would the day before an actual race/competition.
- Wear whatever you are comfortable cycling in; however, a chest heart rate strap will be used and may require us to make adjustments to get a good signal.

Testing will take place in the Human Performance Lab (room 225) in Mitchell Hall on the University of Wisconsin – La Crosse campus. Come into Mitchell through any of the doors on the southeast side (off of Campbell Street) and go up the stairs by the athletic weight room/Mitchell pool to be in the correct hallway; we are located on the far East end. http://www.uwlax.edu/sah/ess/hprl/html/contact.htm
Any other questions should be directed to me at malterer.kath@uwlax.edu or 507-420-5733.

Thank you again and see you soon!
Katherine (Kate) Malterer

APPENDIX D

RISK ASSESSMENT

Are you over the age of …	Yes	No
45 AND a male		
55 AND a female		
Do you have any diagnosed…	**Yes**	**No**
Cardiovascular Disease (cardiac, peripheral, or cerebrovascular disease)		
Pulmonary Disease (COPD, asthma, interstitial lung disease, or cystic fibrosis)		
Metabolic Disease (Diabetes (Type I or II), thyroid disorders, or renal or liver disease		

Please answer all of the following questions as well.

Risk Factor	Description	Yes	No
Family History	Heart attack, coronary revascularization, or sudden death in an immediate relative (male <45 years old or female <55 years old)		
Cigarette Smoking	Current smoker or those who have quit in the last 6 months		
Hypertension (High Blood Pressure)	SBP (top number) ≥140mmHg **OR** DBP (bottom number) ≥ 90mmHg confirmed by measurement on at least 2 separate occasions **OR** on a hypertensive medication		
Dyslipidemia (High Cholesterol)	Total Cholesterol >200mg/dL **OR** HDL <40mg/dL **OR** LDL >130mg/dL (use LDL rather than Total Cholesterol if value is known), **OR** on lipid lowering medication		
Impaired Fasting Glucose	Fasting glucose ≥100mg/dL confirmed on 2 separate occasions		
Obesity	BMI >30 **OR** waist girth >102cm (40in) in men and >88cm (35in) for women **OR** waist-to-hip ratio of ≥.95 for men and ≥.86 for women		
Sedentary Lifestyle	Not participating in a regular exercise program or accumulating 30 minutes or more of moderate activity on most days of the week		
High Serum HDL (positive factor)	>60mg/dL		

Signs or Symptoms	Yes	No
Pain or discomfort in the chest, neck, jaw, arms, or other areas that may be due to myocardial ischemia (lack of adequate circulation)		
Shortness of breath at rest, during activities, or with mild exertion		
Dizziness or syncope (fainting)		
Orthopenea (breathing discomfort when not in an upright position) or paroxysmal nocturnal dyspnea (interrupted breathing at night)		
Ankle edema (swelling)		
Palpitations (abnormal rapid beating of the heart) or tachycardia (rapid heartbeat)		
Intermittent claudication (cramping, pain, and weakness in legs, especially calves, during walking due to inadequate blood supply to muscles)		
Known heart murmur (atypical heart sound indicating a structural or functional abnormality)		
Unusual or unexplained fatigue		

Name_____Signature_____Date_____

_____

APPENDIX E

PRE-PARTICIPATION QUESTIONAIRE

Pre-Participation Questionnaire

For the purpose of validity, it is important that your basic conditioning and health does not change significantly over the course of this study. To account for external variables that may interfere with data results, we would like you to fill out this pre-participation questionnaire before each test.

Read each question carefully and place an "**X**" in on the appropriate line. Please answer honestly and to the best of your knowledge.

If you answered **YES** to any of your questions, please explain below.

Yes	No	
		1. Have you worked out intensely in the last 24 hours?
		2. Have you had any illness or injury in the past 2 days?
		3. Have you significantly changed your exercise habits in the past 2 days?
		4. Have you consumed a significant amount of alcohol in the last 24 hours?
		5. Do you have a current racing license?
		6. Do you know of **any other reason** why you should not do physical activity today?
		7. How many hours of **TOTAL** exercise do you perform each week?
		8. How many hours of cycling training do you currently participate in per week?
		9. How many hours of cycling training did you participate in last month?
		10. How many hours of cycling training did you participate in over the last summer?
		11. How many cycling races do you participate in per year?

Participant _____Signature _____Date

APPENDIX F

REVIEW OF LITERATURE

REVIEW OF LITERATURE

Ambulatory, pursuit type, sports such as running, swimming, cycling, and speed skating depend on a dynamic balance between power output from the muscles and power losses to the environment. Because much of muscular power output is dependent on aerobic metabolism, power output is systematically decreased at higher altitude. However, in air resisted activities such as cycling and speed skating, the reduction in power losses to air friction is so substantial that they are larger than the decreases in aerobic power output (VO_{2max} and/or ventilatory/lactate threshold), with the result that performance time is actually better (e.g. reduced).

Central Governor

The Central Governor is the mechanism Hill, Long, and Lupton (1924) coined to explain the decrease in Q during blood desaturation in hypoxia. This central control receives large amounts of afferent information about various systems or the body and sends efferent instructions on how to adjust the output of energy in the most efficient way. The heart and/or nervous system are controllers of this mechanism with the primary task of maintaining arterial blood saturation (SaO_2) levels in the body. If SaO_2 dropped too low, the controller would reduce Q to maintain as much O_2 in the blood as possible. If and when the SaO_2 began to increase to normal levels, Q would also increase until it reached a homeostatic state.

Ulmer (1996) revisited this idea of a central regulator many years later, but turned his focus to the extracellular feedback of skeletal muscle and its role in optimal

adjustment of energy consumption in runners. He assumed motion, force output, time, and metabolic rate of skeletal muscle would be the most essential components to determine performance and that an integration of these signals would require complex calculations to be performed instantaneously by a control center. These calculations would be based on efferent metabolic rates and exercise intensity in relation to a predicted endpoint and send afferent information through the peripheral and central nervous systems to regulate biomechanics along with the muscular metabolism. This process by the central control system was called "teleoanticipation" and included the idea that rates of energy expenditure might have a behavioral psychopysiological influence.

This idea of a psychological component was not founded by Ulmer, however. Borg (1982) originally created a scale of 6 – 20 called the Rating of Perceived Exertion (RPE) to attempt to quantify feelings of exertion in exercise. Since then, RPE growth has been correlated to increases in muscular force, heart rate, ventilation, respiratory rate, oxygen uptake, and blood lactate levels (Albertus et al., 2005; Borg, 1982; Foster et al., 2009; Hampson et al., 2004; St. Clair Gibson et al., 2006). Environment changes such high heat and hypoxia increase RPE at a more rapid rate and decrease time to exhaustion (Tucker, 2009). Overall RPE and heart rate responses even increase despite external information or misinformation of exercise intensity (Hampson et al., 2004), demonstrating the robustness of RPE in its accuracy of externally describing internal fluctuations from homeostasis. However, the growth of RPE during any event seems to be scaled to the relative length of the event (Faulkner, Parfitt, & Roger, 2008; Joseph et al., 2008).

The CG employs many signals to maintain homeostasis in the body and protect from unnecessary fluctuations (Noakes, St. Clair Gibson, & Lambert, 2005; Lambert, St. Clair Gibson, & Noakes, 2005). Peripheral fatigue is described as down regulated force production and PO from reduced muscle contractility caused by physiological changes (Hettinga, de Koning, Broersen, Geffen, & Foster, 2006). Metabolic substrates, including glucose, Ca^{2+}, lactic acid, and muscle glycogen, are likely signalers of peripheral fatigue rather than by-products. These substrates, along with the negative feedback of SaO_2 levels, blood glucose concentration, acid-base balance, arterial blood pressure, core temperature, and plasma osmolality were thought to work independently towards the goal of signaling the central nervous system to bring the body back to homeostasis or at least to prevent unreasonably large homeostatic disturbances. Excessive afferent feedback from high initial work rates signalled by these elements would cause premature fatigue and a rapid reduction in PO as well (Tucker, 2009).

When combining these physiological feedbacks with teleoanticipation, the CG became a more objective regulator of energy expenditure rather than singularly cognitive as was postulated in Ulmer's (1996) work. However, teleoanticipation did have the ability to "reset" or "hardwire" the CG with training. In addition, it was supported that psychological factors become less important at high exercise intensities with afferent sensations becoming the primary signaler of effort perception and fatigue (Hampson et al., 2004).

Noakes and colleagues (Noakes, Peltonen, & Rusko, 2001) also looked to support the CG theory in regards to its specific function of protecting the heart and preventing myocardial ischemia. Instead of reduced Q, heart rate maximum (HR_{max}), and the VO_{2max}

being the cause of a reduced skeletal muscle PO, it would be a result of the "governor" down regulating the body's workload to maintain SaO_2 levels which prevent myocardial ischemia and cerebral hypoxia in maximal exercise. Reduced PO would decrease the action of the muscle pump which, in turn, would decrease venous return, thus explaining a lower Q and preventing catastrophic homeostatic disturbance (Noakes et al., 2001).

Thus it has been shown that perception of effort and fatigue are not correlated with just one peripheral variable (St. Clair Gibson et al., 2006), but are a continual conversation from peripheral information to central control throughout the body.

Hypoxia

At a given oxygen uptake level (VO_2) or submaximal load, heart rate (HR), and skeletal muscle blood flow are increased to maintain oxygen availability close to homeostasis (Noakes et al., 2001) as oxygen (O_2) transport to the muscles is the principle limiter in VO_{2max} (Amann, 2011; Noakes et al., 2001). However, during maximal effort in hypoxia, VO_{2max} is reduced (Mollard et al., 2007; Noakes et al., 2001) along with decreases in HR_{max} and maximal stroke volume thus causing a lowering of cardiac output (Q) (Noakes et al., 2001; Peltonen, Tikkanen, & Rusko, 2001). If Q would continue to increase, as it does in hypoxic submaximal work, a detrimental arterial desaturation would occur (Peltonen et al., 2001). These distinct reductions in HR_{max} and stroke volume are seen with exercise despite increased sympathetic responses such as blood lactate and noreprinephrine concentrations which should upregulate these functions instead of down regulate (Noakes et al., 2001).

The reduction in Q and, subsequently, the VO_{2max} as a result of decreased peripheral work in hypoxia with a delay in conscious perceived effort futher supports the

CG theory (Johnson et al., 2009; Peltonen et al., 2001). Perceptions of effort are increased for both respiration and the overall body while absolute VO_2 remains the same at a given workload (Amann, 2011; Noakes et al., 2001) and are more or less severe depending on the characteristics of the task being performed (Amann, 2011).

This reduction in locomotor muscle power output (PO) is likely a combination of central and peripheral signals as suggested by the Central Governor (CG) theory. The rate of peripheral locomotor muscle fatigue, which is regulated by O_2 transport to the working muscles, is used as a "dose dependent" trigger for reduced central motor output (Amann et al., 2006; Millet, Muthalib, Jubeau, Laursen, & Nosaka, 2012). This rate is accelerated by the accumulation of fatigue metabolites, increased type II muscle recruitment, and metabolic acidosis (Amann, Pegelow, Jacques, & Dempsey, 2007) with an increase in the work and subsequent fatigue of respiratory muscles acting as a trigger on the effects of the sympathetic nervous system, vascular resistance, and blood flow in locomotor muscles (Amann et al., 2006; Amann et al., 2007). However, in cases of whole body exercise, cerebral hypoxia may signal PO reductions before peripheral fatigue becomes substantantial enough to reduce exercise capacity (Amann et al., 2007) and especially in highly intense exercise or extreme hypoxia (Millet et al., 2012). The robustness of this feedback system is supported by large decreases in PO with only small changes in O_2 concentrations (Amann, 2011; Peltonen et al., 2001), even as small as 10% or less (Amann et al., 2006) and a linear increase of perceived exertion even with significant reduction in PO (Johnson et al., 2009). In fact, absolute values and rate of rise of ratings of perceived exertion in hypoxia continued increasing when compared with normoxic conditions at the same work rate and duration (Romer et al., 2007).

For all humans in hypoxia, there continues to be a pronounced "reserve of energy" at the end of time trials as predicted by the CG theory (Hampson et al., 2004; Jaime et al., 2012). Though the level and rate of development of peripheral fatigue are suggested to be the primary markers for central motor output decrease for work in hypoxia (Romer et al., 2007), exercise time to exhaustion is 70% in hypoxia compared to normoxia without a significant increase in peripheral fatigue, blood lactate concentrations, and relative force output at the point of exhaustion in both conditions (Romer et al., 2007). This is attributable to a more rapid reduction in Q and increase in muscle recruitment and lactate accumulations values signalling the cessation of exercise with a physiological reserve at altitude (Hampson et al., 2004).

Pacing Strategies

Pacing strategies can be defined as the distribution of energy to maximize performance and minimize homeostatic disturbance over a given distance or time. The strategy is based on a complex combination of peripheral and central feedforward/feedback mechanisms (Abbiss & Larson, 2008; de Koning & et al., 2011; Foster & et al., 2012; Hill et al., 1924; Noakes et al., 2005; Noakes et al., 2001; St. Clair Gibson et al., 2006). Several specific pacing strategies had been previously defined by Abbiss and Larson (2008) and include the following: Negative Pace, where PO is low in the beginning and increases throughout the event.; Positive Pace, where PO is maximal at the start of the event and decreases linearly; All Out Pace, where PO is maximal the entire duration; and Even Pace, where PO is relatively constant throughout the event.

The initial selection of each pacing strategy, known as the pre-race template (Foster et al., 2009), is based on the perceived distance or time to completion of the event

by each individual competitor. However, after the event begins, internal and external disturbances will influence whether the pacing pattern will stay the same or change (Foster et al., 2009). Fluctuation of pace can lessen with practice, as seen with athletes reproducing exercise intensity when using only Rating of Perceived Exertion (RPE) to moderate intensity (St. Clair Gibson et al., 2006).

Distances of 3km to 5km as seen in the 1980 and 1988 Olympic cycling competitions (Foster et al., 1993) initially seemed to have an Even Pace that was later found to not be so even. Instead, it was a Parabolic Pace or Variable Pace which starts as a Positive Pace, changes to an Even Pace in the middle, and finishes as a Negative Pace at the end of the trial (Abbiss & Larson, 2008; de Koning, Bobbert, & Foster, 1999; St. Clair Gibson et al., 2006). The optimal use of this Parabolic Pace is an "All Out" start of approximately 12 seconds with a switch to an Even Pace for the remainder of the event in a theoretically perfect race; however, true Olympic cyclists always reserved enough energy for an end "kick" (de Koning et al., 1999). This kick was likely a reserve of energy that the body held as a back-up to prevent a catastrophic homeostatic disturbance in self-paced time trials (Crewe, Tucker, & Noakes, 2008; St. Clair Gibson et al., 2006; Mauger, Jones, & Williams, 2011) and to prevent exhaustion before the completion of the race (Ulmer, 1996). This reserve has been found in other, non-elite athletic settings and is especially large when learning a new task or having no knowledge of an endpoint (Mauger et al., 2011). This is hypothesized to be because the CG cannot accurately select a set pacing strategy and must heavily rely on peripheral feedback to regulate PO (Mauger, Jones, & Williams, 2009). However, when an individual has had sufficient practice and nears the end of an exercise bout, the level of uncertainty that they will

finish the race diminishes, allowing them to utilize the remaining motor unit recruitment and metabolic reserve (St. Clair Gibson et al., 2006; Tucker, 2009).

Pre-race template

The pre-race template is created with the knowledge of an endpoint of the task (like the finish line in a race) along with similar, prior experiences (St. Clair Gibson et al., 2006). Just as the CG is able to be "reset" or "hard-wired" based on previous experiences and training (Lambert et al., 2005), the pre-race template is modified with experience (St. Clair Gibson et al., 2006). Ansley, Robson, St. Clair Gibson, and Noakes (2004) showed that, when given a set endpoint, the body can accurately pick a PO that is suitable to fit that distance. However, when that distance changes, as it did when the researchers deceived the participants into cycling a 33s and 36s Wingate Anaerobic Test (instead of the standard 30s), the body's peripheral signals will caused a lowering of PO even though the subject was not consciously aware of the miscalculation. Thus, the template held constant until it hit an absolute need to change the pacing strategy. The template also has an RPE component where an athlete's expectation of effort is selected prior to the upcoming task and is also used as a gauge to modify work within the event with knowledge of an endpoint (Tucker, 2009).

However, the pre-race template is robust and not always easily changed. This has been shown by Jaime et al. (2012) when cyclists performed normoxic time trials with a normoxic warm-up and later did hypoxic time trials with hypoxic warm-ups. Initial PO in both cases was the same, so even though afferent feedback for hypoxia was signalling to reduce PO already in the warm-up, the template was still selected for the normoxic conditions which the subjects were most habituated to from living at sea level.

Conclusion

The process of interpreting peripheral feedback is done by a "central governor" which uses received information about peripheral fatigue to signal disturbances in the body's homeostatic state and adjust to a pacing strategy that is better suited to help an individual reach a defined endpoint. This control system seeks to navigate the pacing landscape which encompasses all pacing variations that the individual has experienced at a particular exercise duration and intensity. Even though the CG and pre-race template are fairly robust, they still adapt to changes in external environmental changes and internal homeostatic disturbances by adjusting PO. In hypoxia, a change in PO is expected to help maintain SaO_2 levels, however, even with a hypoxic warm-up the initial PO is still the same as it is in normoxia, thus showing a major contraindition. This high initial PO at hypoxia needs to be compensated for later in the race, possibly by the lower final PO. However, this hypoxia research has traditionally been conducted by comparing normoxic maximal test values with hypoxic experimental trial values and assuming that the same mechanisms that cause PO decreases in normoxia cause the same homeostatic disturbances in hypoxia, though they are known to be different. Therefore, by studying TT PO in both hypoxic and normoxic conditions and comparing them to their maximal PO in each respective condition, we can better understand the true PO at each condition.

References

Abbiss, C. R., & Laursen, P. B. (2008). Describing and understanding pacing strategies during athletic competition. *Sports Medicine*, 38(3), 239-252.

Albertus, Y., Tucker, R., St. Clair Gibson, A., Lambert, E. V., Hampson, D. B., & Noakes, T. D. (2005). Effect of distance feedback on pacing strategy and perceived exertion during cycling. *Medicine and Science in Sports and Exercise*, 37(3), 461-468.

Amann, M. (2011). Central and peripheral fatigue: Interaction during cycling exercise in humans. *Medicine and Science in Sports and Exercise*, 43(11), 2039-2045.

Amann, M., Eldridge, M. W., Lovering, A. T., Stickland, M. K., Pegelow, D. F., & Dempsey, J. A. (2006). Arterial oxygenation influences central motor output and exercise performance via effects on peripheral locomotor muscle fatigue in humans. *Journal of Physiology*, 573(3), 937-952.

Amann, M., Pegelow, D. F., Jacques, A. J., & Dempsey, J. A. (2007). Inspiratory muscle work in acute hypoxia influences locomotor muscle fatigue and exercise performance of healthy human beings. *American Journal of Physiology: Regulatory, Integrative and Comparative Physiology*, 293, R2036-R204.

Ansley, L., Robson, P. J., St. Clair Gibson, A., & Noakes, T. D. (2004). Anticipatory pacing strategies during supramaximal exercise lasting longer than 30 s. *Medicine and Science in Sports and Exercise*, 36(2), 309-314.

Borg, G. A. U. (1982). Psychophysical bases of perceived exertion. *Medicine and Science in Sports and Exercise*, 14(5), 377-381.

Crewe, H., Tucker, R., & Noakes, T. D. (2008). The rate of increase in rating of perceived exertion predicts the duration of exercise to fatigue at a fixed power output in different environmental conditions. *European Journal of Applied Physiology*, 103, 569-577.

de Koning, J. J., Bobbert, M. F., & Foster, C. (1999). Determination of optimal pacing strategy in track cycling with an energy flow model. *Journal of Science and Medicine in Sport*, 2(3), 266-277.

de Koning, J. J., Foster C., Bakkum A., Kloppenburg S., Thiel C., Joseph T.,... Porcari J. P. (2011). Regulation of pacing strategy during athletic competition. *PLoS ONE*, 6(1), 1-5.

Faulkner, J., Parfitt, G., & Roger, E. (2008). The rating of perceived exertion during competitive running scales with time. *Psychophysiology*, 45(6), 977-985.

Foster, C., de Koning, J. J., Bischel, S., Casolino, E., Malterer, K., OBrien, K.,... Van Tunen, J. (2012). Pacing strategies for endurance performance. In I. Mujika (Ed.), *Endurance training science and practice* (1 ed., Vol. 1, pp. 85-98). Trapagaran, Bizkaia: Samper Impresores.

Foster, C., Hendrickson, K. J., Peyer, K., Reiner, B., de Koning, J. J., Lucia, A.,… Wright, G. (2009). Pattern of developing the performance template. *British Journal of Sports Medicine*, 43, 765-769.

Foster, C., Snyder, A. C., Thompson, N. N., Green, M. A., Foley, M., & Schrager, M. (1993). Effect of pacing strategy on cycle time trial performance. *Medicine and Science in Sports and Exercise*, 25(3), 383-388.

Hampson, D. B., St. Clair Gibson, A., Lambert, M. I., Dugas, J. P., Lambert, E. V., & Noakes, T. D. (2004). Deception and perceived exertion during high intensity running bouts. *Perceptual and Motor Skills*, 98, 1027-1038.

Hettinga, F. J., de Koning, J. J., Broersen, F. T., Geffen, P. V., & Foster, C. (2006). Pacing strategy and the occurrence of fatigue in 4000-m cycling time trials. *Medicine and Science in Sports and Exercise*, 38(8), 1484-1491.

Hill, A. V., Long, C. N. H., & Lupton, H. (1924). Muscular exercise, lactic acid and the supply and utilization of oxygen. *Proceedings of the Royal Society: Biological Sciences*, 97, 155-176.

Jaime, C., Nyberg, K., Condello, G., Casolino, E., Dodge, C., Marroyo-Rodriguez, J.,… Foster, C. (2012). *The effect of manipulating the pre-exercise template on time trial performance*. In Meeusen, R., Duchateau, J., Roelands, B., Klass, M., De Geus, B., Baudry, S., Tsolakidis, E. (Eds.), *17th annual congress of the European college of sport science book of abstracts* (p. 74).

Johnson, B. D., Joseph, T., Wright, G., Battista, R. A., Dodge, C., Balweg, A.,… Foster, C. (2009). Rapidity of responding to a hypoxic challenge during exercise. *European Journal of Applied Physiology*, 106, 493-499.

Joseph, T., Johnson, B., Battista, R. A., Wright, G., Dodge, C., Porcari, J. P.,… Foster, C. (2008). Perception of fatigue during simulated competition. *Medicine and Science in Sport and Exercise*, 40(2), 381-386.

Lambert, E. V., St. Clair Gibson, A., & Noakes, T. D. (2005). Complex systems model of fatigue: integrative homeostatic control of peripheral physiological systems during exercise in humans. *British Journal of Sports Medicine*, 39, 52-62.

Mauger, A. R., Jones, A. M., & Williams, C. A. (2009). Influence of feedback and prior experience on pacing during a 4-km cycle time trial. *Medicine and Science in Sports and Exercise*, 41(2), 451-458.

Mauger, A. R., Jones, A. M., & Williams, C. A. (2011). The effect of non-contingent and accurate performance feedback on pacing and time trial performance in 4-km track cycling. *British Journal of Sports Medicine*, 45(3), 225-229.

Millet, F. Y., Muthalib, M., Jubeau, M., Laursen, P. B., & Nosaka, K. (2012). Severe hypoxia affects exercise performance independently of afferent feedback and peripheral fatigue. *Journal of Applied Physiology*, 112, 1335-1344.

Mollard, P., Woorons, X., Letournel, M., Lamberto, C., Favret, F., Pichon, A.,…Richalet, J. (2007). Determinants of maximal oxygen uptake in moderate acute hypoxia in endurance athletes. *European Journal of Applied Physiology*, 100, 663-673.

Noakes, T. D., Peltonen, J. E., & Rusko, H. K. (2001). Evidence that a central governor regulates exercise performance during acute hypoxia and hyperoxia. *Journal of Experimental Biology*, 204, 3225-3234.

Noakes, T. D., St. Clair Gibson, A., & Lambert, E.V. (2005). From catastrophe to complexity: A novel model of integrative central neural regulation of effort and fatigue during exercise in humans: Summary and conclusions. *British Journal of Sports Medicine*, 39, 120-124.

Peltonen, J. E., Tikkanen, H. O., & Rusko, H. K. (2001). Cardiorespiratory responses to exercise in acute hypoxia, hyperoxia, and normoxia. *European Journal of Applied Physiology*, 85, 82-88.

Romer, L. M., Haverkamp, H. C., Amann, M., Lovering, A. T., Pegelow, D. F., & Dempsey, J. A. (2007). Effect of acute severe hypoxia on peripheral fatigue and endurance capacity in healthy humans. *American Journal of Physiology: Regulatory, Integrative and Comparative Physiology*, 292, R598-R606.

St. Clair Gibson, A., Lambert, E. V., Rauch, L. H. G., Tucker, R., Baden, D. A., Foster, C., & Noakes, T. D. (2006). The role of information processing between the brain and peripheral physiological systems in pacing and perception of effort. *Sports Medicine*, 36(8), 705-722.

Tucker, R. (2009). The anticipatory regulation of performance: the physiological basis for pacing strategies and the development of a perception-based model for exercise performance. *British Journal of Sporst Medicine*, 43(6), 392-400.

Ulmer, H. V. (1996). Concept of an extracellular regulation of muscular metabolic rate during heavy exercise in humans by psychophysiological feedback. *Experientia*, 52, 416-420.